~~Chromosome~~ Chromatin immunoprecipitation assay was performed to further confirm these findings. KLF9 was found to actually bind to the endogenous adiponectin promoter region comprising the 32-bp site (Fig. 4e). Furthermore, purified KLF9 also showed nearly the same inhibition of the 32-bp fragment as that by a nuclear extract prepared from adipocytes or adipose tissue (Fig. 4f).

wf 22310 Please replace the paragraph beginning at page 25, line ~~12~~ 14 with the following:

Next, to study the functional relationship between adiponectin expression and KLF9 *in vivo*, the phenotypes of KLF9 knockout mice were analyzed ~~(Fig. 8a)~~ (Fig. 8) (Morita, M. *et al.*, Mol. Cell. Biol. 23, 2489-2500 (2003)). Interestingly, the 32-bp binding protein was not detected in the nuclear extract derived from KLF9 knockout mice "WAT" ~~(Fig. 8b)~~ (Fig. 8). Importantly, despite that the weight of KLF9 knockout mice was lower than that of the control wildtype littermates, the plasma adiponectin level in KLF9 knockout mice was lower than that of the control wildtype littermates ~~(Fig. e)~~ (Fig. 8). In contrast, no difference in the plasma adiponectin level was observed between KLF3 knockout mice and their control wildtype littermates (data not shown). These data suggested that KLF9 plays an important role in the regulation of the adiponectin level *in vivo*.

Please replace the paragraph beginning at page 26, line 10 with the following:

To further confirm the above-mentioned hypothesis, the change in oxidative stress during adipocyte hypertrophy was measured. Genomic DNAs extracted from 3T3L1 cells (day 10 and day [[18]]19) were degraded, and the amount of 8-OHdG (oxidized form of dG) in the degradation products of genomic DNA was measured by ELISA using a specific antibody. The amount of 8-OHdG increased along with differentiation (Fig. 10). The change of antioxidative activity that accompanies adipocyte hypertrophy was